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# Vestibular hyperreactivity and hyperventilation after whiplash injury

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## Abstract

Vestibular, oculomotor and respiratory tests were performed on 32 patients after whiplash injury caused by a rear-end car collision. Oculomotor functions were generally normal. The cervico-ocular reflex was usually absent or displayed the low gain typical of normal subjects. There was no nystagmic response to static neck torsion. The vestibulo-ocular reflex showed vestibular hyperreactivity (VH) in a significantly large number of cases ( $n = 17$ ; 53%). The respiratory test results were also typical of the hyperventilation syndrome (HVS) in a significantly large number of cases ( $n = 12$ ; 38%). The findings of VH and the HVS were not significantly correlated within the patient group. However, the combination of VH and the HVS occurred significantly more often ( $n = 7$ ; 22%) than could be accounted for by combined false positivity. Most of the significant findings were due to high relative frequencies in the women: 11 out of the 17 women (65%) showed VH, 8 (47%) had the HVS and 5 (29%) showed a combination of VH and the HVS. The findings were not correlated with the patient's age or the time interval between the accident and the examination. VH might have been the result of plastic adaptation to limited head mobility secondary to neck pain. Behavioural and emotional distress might offer alternative explanations for both VH and the HVS.

**Keywords:** Vestibulo-ocular reflex (VOR); Cervico-ocular reflex (COR); Emotional disorder; Smooth Pursuit; Optokinetic nystagmus

## 1. Introduction

It has been estimated that about 10% (Chester, 1991) to 20% (Toglia, 1976) of the occupants of the stricken vehicle in rear-end car collisions suffer whiplash injury. This injury is defined as a non-contact acceleration-deceleration injury to the head-neck system. It is most often caused by a rear-end car collision and there is no direct impact on the neck. The biomechanics of whiplash injury are complex. Presenting symptoms usually include neck pain, headaches, disequilibrium, blurred vision, paraesthesiae, changes in cognition, fatigue, insomnia and hypersensitivity to light and sound (Bogduk, 1986; Zenner, 1991). Imbalance, light-headedness and vertigo also occur frequently and

these symptoms may be associated with long-term disability. However, there is considerable controversy as to whether whiplash injury gives rise to any relevant objective otoneurological abnormalities. Several have been implicated (see Discussion) such as abnormalities of the oculomotor reflexes, the vestibulo-ocular reflex (VOR) and the cervico-ocular reflex (COR). In a number of consecutive cases who we have examined after whiplash injury over the past few years, the results of the velocity step (VS) test demonstrated vestibular hyperreactivity (VH) of the VOR (see Discussion). In view of our previous work on the hyperventilation syndrome (HVS) (Theunissen et al., 1986) and the similarities between the symptoms of whiplash injury and hyperventilation, we decided to actively recruit a specific group of patients for a controlled study. Suitable cases were asked to complete a standard questionnaire about the accident and their injuries. After enrolment they were scheduled for tests according to a

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standard protocol. The purpose of this study was to (1) evaluate the occurrence of VH in whiplash patients, and (2) investigate possible relationships between whiplash injury and the HVS. Part of this study is reported in abbreviated form (Fischer et al., 1995).

## 2. Subjects and methods

Vestibular, oculomotor and respiratory function tests were performed on 32 patients after whiplash injury (15 men, 17 women, age range 21–66 years) caused by a rear-end car collision 1–26 months previously. They were referred to us (TF, ET) by general practitioners from a local region who had been informed about this study. Before enrolment, the potential participants completed a questionnaire (Zenner, 1991) to check whether the collision and injury fulfilled our study criteria. These criteria concerned only the type of accident and injury and disregarded the symptoms and/or signs. Candidates who reported any other relevant disorder or who were using any relevant drugs, were excluded from the study. Each participant underwent routine diagnostic, radiodiagnostic (standard and functional X-rays of the cervical spine) and otoneurological tests.

Apart from the present group of whiplash patients, we also studied a small group ( $n = 7$ ) of patients who had suffered (non-contact) injuries caused by a non-rear-end car collision. In addition, some of the relevant results obtained from a different group of consecutive patients with whiplash injuries ( $n = 35$ ) who had been referred routinely to us (PH) were compared to the present findings (see Discussion).

### 2.1. Oculomotor tests

Eye movements were recorded with direct-current electro-oculography (EOG). Calibration of eye movement was performed before each test by having the patient look in alternation at two light dots  $10^\circ$  on either side of the primary position. Gaze positions were tested to see whether there was any gaze-evoked nystagmus (in light, the patient fixated a target at about  $30^\circ$  to  $40^\circ$  lateral displacement or  $20^\circ$  vertical displacement). Saccades, smooth pursuit (SP) and optokinetic nystagmus (OKN) responses were elicited and analyzed as previously reported (Verhagen et al., 1992). The SP stimulus was a sinusoidal movement at 0.33 Hz with  $20^\circ/\text{s}$  peak velocity. The two velocities used for the OKN stimulus (shadow stripes, width and separation  $7.5^\circ$ , projected onto a hemicylindrical screen) were 40 and  $60^\circ/\text{s}$ . An average slow phase velocity (SPV) of nystagmus was derived for each OKN response from the EOG record. For the OKN responses, age-related 95% confidence limits were used which had been obtained with a regression analysis (average SPV on age) on 75 control subjects of various ages.

### 2.2. Cervico-ocular reflex (COR) tests

The COR was elicited with the patient seated in an upright position on the rotatory chair and the head fixed in space (by the examiner's hands, under close-up infrared video monitoring control). The body was rotated sinusoidally under the fixed head with a  $60^\circ$  peak-to-peak amplitude at the frequency 0.1 Hz ( $19^\circ/\text{s}$  maximum velocity). The COR was evaluated in total darkness with the eyes open. The patient received no instructions other than to relax. The position of the eyes was monitored by using an infrared video camera. The gain was measured from the EOG using the maximum SPV (MSPV) and cumulative (fast phase) eye displacement (CED) per hemicycle as previously described (Huygen et al., 1991). In addition, a ramp/plateau stimulus was used according to Holtmann et al. (1989). A  $5^\circ/\text{s}$  ramp (angular) displacement was applied during 6 s (amplitude  $30^\circ$ ) and, after a pause of about 10 s (at a fixed angular displacement of  $30^\circ$ ), a similar ramp displacement was executed back to the primary position, which was followed by an additional recording interval of some 10 s. Two consecutive tests took place with the first ramp displacements in alternating directions. For a qualitative evaluation, we judged whether nystagmus, other than pre-existent nystagmus, could be observed during the plateau phase ( $30^\circ$  static neck torsion). The MSPV was used as a quantitative response parameter.

For the sinusoidal COR stimulus, the 95% confidence limits were derived from the means and the SDs of the gains at 0.1 Hz previously reported for a group of normal subjects (Huygen et al., 1991). For the ramp COR stimulus, we had no normal values of our own so we derived a tentative P95 value of  $6^\circ/\text{s}$  for a ramp velocity of  $5^\circ/\text{s}$  (note that this corresponds with a velocity gain of 1.2) from a previous study (Holtmann et al., 1989) in which the equipment and conditions were essentially the same as ours.

### 2.3. Vestibular tests

Vestibular tests were conducted with the patient in the dark with the eyes open. It was checked whether there was any spontaneous nystagmus ( $\text{SPV} > 7^\circ/\text{s}$ ). VS tests were performed using a rotatory chair (Tönnies, Freiburg im Breisgau, Germany). After  $0.8^\circ/\text{s}^2$  acceleration and an interval of  $90^\circ/\text{s}$  constant velocity long enough to allow the perrotatory nystagmus response to subside, the chair was stopped at a deceleration of  $200^\circ/\text{s}^2$ . The postrotatory nystagmus response was analyzed using a computer (Theunissen et al., 1986, 1988). The following response parameters were used to characterize the VOR: initial velocity ( $V$ , 90% confidence limits 30 to  $65^\circ/\text{s}$ ), time constant ( $T$ , 11 to 26 s) and "Gesamtamplitude" or cumulative eye displacement ( $G = VT$ , 485 to  $1135^\circ$ ). The criterion for VH was that at least 1 response parameter had a value (for either nystagmus direction) of higher than the



upper confidence limit ( $p = 0.05$  for each single parameter for each nystagmus direction). As previously reported (Theunissen et al., 1986), the percentage of (overall) false positivity using this criterion is somewhere in the range of between 14 and 26%. To calculate the directional preponderance (DP) for each of the parameters V, T and G, we used a formula which was similar to that of the side effect (see below). An average of 25% was used as a 95% confidence limit for these DP values (Theunissen et al., 1986). If the VS responses for the two nystagmus directions were asymmetric, the caloric test was performed. In this test, the response parameter was the maximum SPV of nystagmus at the culmination of the response. For the side effect (SE), it was arbitrarily assumed that the relative difference between both sides, i.e.  $SE = (\text{difference}/\text{sum})$  100% in excess of 20% indicated unilateral hypofunction. The 95% confidence limits for the absolute response level after 20 s irrigation with (150 cm<sup>3</sup>) water at 30°C and 44°C, were 7°/s and 45°/s (Nijhuis and Huygen, 1980). Irrigations at 44°C were only performed if there was a considerable difference between the cold caloric responses from both sides and/or there was a significant DP found in the VS responses.

#### 2.4. Respiratory tests

The diagnosis of the hyperventilation syndrome (HVS) was based on the results obtained during a ventilatory response to CO<sub>2</sub>, a hyperventilation provocation test and from arterial blood gas values. The ventilatory response to CO<sub>2</sub> was measured by adding 2–3% CO<sub>2</sub> to the inspiratory air. The hyperventilation provocation test consisted of 1 min of voluntary overbreathing. The end-tidal PCO<sub>2</sub> was measured continuously with a capnograph before, during and after hyperventilation. Positive indications for the HVS were: a decrease in ventilation when enough CO<sub>2</sub> was given in the inspiratory air to increase the end-tidal PCO<sub>2</sub> by 0.7 kPa (5 torr); high frequency of ventilation; irregular breathing pattern; recognition of symptoms during hyperventilation provocation; slow recovery of alveolar PCO<sub>2</sub> after the provocation and arterial hypocapnia. The diagnosis of the HVS was made if at least 3 of the above features were observed. A negative base excess (> 2 mM/l) was considered to be an indication of a chronic HVS (Folgering and Colla, 1978).

#### 2.5. Statistical analysis

Individual parameter values obtained on all the tests were compared to the corresponding above-mentioned normal values. Differences between the relative frequencies of any feature were tested in a 2 × 2 contingency table using Fisher's exact probability test at a 5% probability level. The resulting  $F$  (cf.  $p$  value) is specified only for significant differences (i.e.,  $F < 0.05$ ). The  $\chi^2$  test was employed for sufficiently high separate frequencies. A signifi-

Table 1

Minimum number of occurrences given for the binomial distribution with sample size  $n$  and chance of occurrence ( $p$ ) for which the tail probability  $p$  is  $< 0.05$ . For example, 12 out of 32 patients with the HVS was significantly high because  $< 5$  patients could be expected on the basis of false positivity ( $p = 0.05$ ).

$n$	$p$		
	0.007–0.013 <sup>a</sup>	0.05 <sup>b</sup>	0.14–0.26 <sup>c</sup>
32–35	2–3	5	9–14
17	2–2	4	6–8
15	2–2	3	5–8
7	1–2	2	4–5
5–6	1–2	2	3–4
3–4	1–1	2	3–4
2	1–1	2	2–4

<sup>a</sup> Whatever type of VH and the HVS in combination.

<sup>b</sup> Single VS response parameters, caloric test, COR test, HVS.

<sup>c</sup> Whatever type of VH (any VS response parameter, any nystagmus direction).

cantly high relative frequency for any relevant feature was detected by determining the tail probability ( $P$ ) of the binomial distribution with the appropriate sample size ( $n$ ) and a value of  $p = 0.05$  (i.e., the chance that this feature would occur). The value of  $p = 0.05$  (i.e. 5% false positivity) applied to the single parameters of the VS test (i.e. for each nystagmus direction), the caloric test, the COR test and the combined criteria for the HVS. In general, a value of  $0.14 < p < 0.26$  applied to VH, as indicated above. For the combination of VH and the HVS, the probability was therefore between  $(0.05)(0.14) = 0.007$  and  $(0.05)(0.26) = 0.013$ . The relevant sample sizes and probabilities plus the minimum frequencies which corresponded with a significantly low tail probability are presented in Table 1.

### 3. Results

#### 3.1. General, oculomotor and COR findings

No gaze-evoked nystagmus was observed and saccades, SP and OKN responses were normal. One patient showed incidental double saccadic pulses (during calibration) (i.e., pairs of small back-to-back saccades without an intersaccadic interval). The sinusoidal COR responses were within

Table 2

Distribution of the VS response parameter values in men and women between and beyond relevant percentiles. Significantly high frequencies are printed in bold.

Confidence limit(s)	R nystagmus			L nystagmus			any V, T or G
	V	T	G	V	T	G	
< P5	3	1	2	1	0	0	3
P5–P95	23	25	21	25	26	19	12
> P95	<b>6</b>	<b>6</b>	<b>9</b>	<b>6</b>	<b>6</b>	<b>13</b>	<b>17</b>
total	32	32	32	32	32	32	32

Table 3  
Same as Table 2: Men

Confidence limit(s)	R nystagmus			L nystagmus			any V, T or G
	V	T	G	V	T	G	
< P5	3	1	2	0	0	0	3
P5–P95	9	13	10	14	14	10	6
> P95	3	1	3	1	1	5	6
Total	15	15	15	15	15	15	15

the normal range for all of the patients, except for one, who had a MSPV gain of between 0.37 and 0.47 and a CED gain of 0.73; the latter was outside the normal range (Huygen et al., 1991). Another patient showed a high-gain COR which consisted mainly of compensatory saccades. Nystagmic responses to the ramp/plateau COR stimulus were only observed during the ramp phases. The measurement of the MSPV gain in these phases added nothing to the gain measurements using sinusoidal stimulation; it should be noted that none of the ramp responses showed a MSPV value of higher than 6°/s. If any response to the ramp was present at all, it was less clear than in the sinusoidal test.

3.2. Vestibular findings

None of the patients showed any spontaneous nystagmus. Caloric responses were normal in all instances, except for one case of bilateral weakness (maximum SPV 5 and 6°/s) and one case of unilateral weakness (6°/s maximum SPV) with a SE of 40%. The VS responses showed a significant DP (31% for V) in only one case. VH was found in 17 patients (53%), which was statistically significant. Separate (relative) frequencies of significantly high V, T, or G values for any nystagmus direction were significantly high in all instances; the highest frequency was observed for G (Table 2). Table 3 and Table 4 show that most of the relatively high frequencies were due to the women's results, as 65% of them showed VH. Nevertheless, we found no significant difference in the relative frequency of VH between the men (Table 3) and the women (Table 4). We could not find any significant correlation between the relative frequency of VH and either the patient's age or the time interval between the accident and the examination.

Table 4  
Same as Table 2: Women

Confidence limit(s)	R nystagmus			L nystagmus			any V, T or G
	V	T	G	V	T	G	
< P5	0	0	0	1	0	0	0
P5–P95	14	12	11	11	12	9	6
> P95	3	5	6	5	5	8	11
Total	17	17	17	17	17	17	17

< /TB4 >

Table 5  
Vestibular hyperreactivity versus the respiratory function test result of the men and women < TB5 >

	Respiratory test result		
	Normal	Typical of HVS	Total
Vestibular Hyperreactivity			
No	10	5	15 (47%)
Yes	10	7 (22%)	17 (53%)
Total	20 (62%)	12 (38%)	32 (100%)

< /TB5 >

3.3. Respiratory findings

The HVS was diagnosed in a significant number of patients: 12 (38%). Chronic HVS was present in 1 of these cases. There was no significant correlation between VH and the HVS within this group. However, it is obvious that the relative frequency of 7 out of 32 (22%) patients with a combination of VH and the HVS is significantly higher than would generally have been expected on the basis of false positivity (Table 1 and Methods). Table 5 Table 6 Table 7 show that there was a significantly high relative frequency of both the HVS and a combination of VH and the HVS in both the men and the women. Almost half (47%) of the women had positive hyperventilation test results and almost 1/3 (29%) showed this feature in combination with VH (Table 7).

3.4. Symptoms and other findings

Only one out of the 31 patients (1 did not complete the questionnaire) was almost symptom-free, while 24 mentioned dizziness. We could not detect any significant difference in the relative frequency of any of the findings between the groups of patients with and without dizziness. However, all 7 of the patients without dizziness were examined within 7 months of the accident, while only 14 out of the 24 patients with dizziness were examined so soon afterwards; this difference was significant ( $F = 0.044$ ).

There was no significant difference in the relative frequency of any of the findings or the presence or absence of the symptom of dizziness between the groups of patients who were or were not using a safety belt, or whose seat had or had not been fitted with a head rest.

Table 6  
Same as Table 5: Men

Vestibular hyperreactivity	Respiratory test result		
	Normal	Typical of HVS	Total
No	7	2	9 (60%)
Yes	4	2 (13%)	6 (40%)
Total	11 (73%)	4 (27%)	15 (100%)



Table 7  
Same as Table 5: Women

Vestibular hyperreactivity	Respiratory test result		
	Normal	Typical of HVS	Total
No	3	3	6 (35%)
Yes	6	5 (29%)	11 (65%)
Total	9 (53%)	8 (47%)	17 (100%)

### 3.5. Non-rear-end car collision patients

Seven patients (3 men and 4 women, aged 20–53 years) underwent all the examinations 2–15 months after their accident (1 man did not complete the respiratory tests). The VS test showed high V values (2 women) and high G values (2 men, 2 women) in one or two nystagmus directions. The HVS was seen in all of the 6 cases tested and was combined with VH in 3 of them (1/2 men, 2/4 women). The finding of VH in 4/7 cases indicates that a comparable proportion of these patients showed a similar type of vestibular disinhibition as that observed in our whiplash patients. The relative frequency of 3/6 cases with a combination of VH and the HVS did not differ significantly from the 7/32 found in our group of whiplash patients with rear-end car collisions or from the relative frequency of about 3/4 cases with this combination in a group of patients who were selected on the basis of the HVS (Theunissen et al., 1986).

## 4. Discussion

### 4.1. General

It was not always possible to make a meaningful comparison between the present results and those of other studies because in many reports the type of accident was insufficiently specified and no mention was made of whether other types of injury or the use of any drugs had been excluded. For example, acute and chronic patients, symptomatic and nonsymptomatic cases, men and women, were not distinguished. The examination methods used by others were generally different from ours and no quantitative and statistical evaluations were mentioned in the majority of the reports.

### 4.2. Oculomotor system

None of our whiplash patients showed any gaze-evoked nystagmus. Saccades and SP/OKN responses were normal in all of them. The only possibly central oculomotor disturbance encountered in the present study was the incidental occurrence of double saccadic pulses (back-to-back saccades) in one patient. Hotson (1982) reported that this phenomenon also occurs in normal subjects. Chester (1991)

found oculomotor abnormalities in only 2 out of 29 chronic patients tested; one patient showed fixation instability and another showed saccadic SP responses. In contrast to these findings, Hildingsson et al. (1989) found a significant reduction in saccade peak velocity and SP gain in a subgroup of 20 chronic patients (who all had severe symptoms of neck pain, neck stiffness and headaches); such abnormalities were lacking in a group of nonsymptomatic patients ( $n = 19$ , interval  $> 6$  months). Rear-end car collisions were involved in only 8 and 10 patients in these two groups, respectively. Oosterveld et al. (1991) reported unilateral gaze-evoked nystagmus in 6%, bilateral gaze-evoked nystagmus in 26% and saccadic SP responses in 43% of the (chronic symptomatic) cases (after unspecified accidents). Neither of the latter two reports mentioned the use of any drugs by the (chronic) symptomatic patients, although it is not unusual for such patients to receive medication which may influence oculomotor functions or cause (gaze-evoked) nystagmus (Pearson and Barber, 1973). Hinoki (1985) mentioned that “abnormal optokinetic nystagmus is often observed” in chronic symptomatic patients. The abnormality was “irregularity” and we assume that the responses showed episodes with a (very) low gain. Ushio et al. (1971), as cited by Hinoki (1985), reported inverted OKN responses after whiplash injury, which seems a peculiar finding unless it was due to pre-existing congenital nystagmus or the superposition of (other) spontaneous or gaze-evoked nystagmus. In patients with head injuries, gaze-evoked nystagmus was reported in less than 1% (Meran et al., 1978) or in about 2% after contusion (Lange and Kornhuber, 1962); impaired SP/OKN responses were found in less than 1%. (Lange and Kornhuber, 1962). Meran et al. (1978) mentioned impaired SP/OKN responses in 10%, especially within the first 6 months. Similar abnormalities were reported in 35–43% of the cases soon after the trauma, which decreased to 8–12% later on (Tuohimaa, 1978; Vartiainen et al., 1985).

### 4.3. COR

Only one patient showed an abnormal response to sinusoidal COR stimulation. He had a significantly high (CED) gain, similar to the values found in labyrinthine-defective subjects (Huygen et al., 1991), but he had normal caloric and rotatory responses; this is not beyond the 5% probability level (i.e. false positivity). A high-gain COR consisting of compensatory saccades can be found in any normal subject, especially when the subject focuses on the movement of the body (Huygen et al., 1991). In the ramp/plateau COR test, none of our whiplash patients showed any nystagmic response to the (30°) static neck torsion. This agrees with the findings previously reported for women (only) (Kingma et al., 1992), using similar equipment (including infrared video recording) and conditions (although with a displacement amplitude of 60°). Our



conclusion is that the ramp/plateau COR test was nonproductive, because the static neck torsion did not produce any relevant response, whereas the nystagmic responses to the 5°/s ramps were unsuitable for distinguishing between normal and “abnormally high” COR gains; this could be done in a reliable way with the sinusoidal COR test.

Oosterveld et al. (1991) reported “cervical nystagmus” in 79% of the patients (with the eyes closed). Unfortunately, the precise stimulus conditions were not specified and control values or statistical analysis were not reported. Normal subjects may show wide normal ranges of their COR activity. Our own measurements (Huygen et al., 1991) indicated that normal gains were in the range of between 0 and 0.6. It is difficult, therefore, if not impossible, to distinguish “positive” and “negative” responses in a valid way without statistical testing. A stimulus amplitude of 60° was used by Oosterveld et al. (1991), although they reported that they met with “restriction of the rotation ability of the head in one or two directions” in 28% of the patients (which instigated our choice of an amplitude of only 30°). It was stressed by both Hülse (1982) and Reker (1983) that in the case of (bi)laterality of the “cervical nystagmus”, the possibility of arousal of any “latent” spontaneous nystagmus should be considered. Interestingly, spontaneous nystagmus was shown by the majority of the patients described by Oosterveld et al. (1991) (see below) and many of their patients (also) showed gaze-evoked nystagmus (see above), whereas these authors did not describe how they distinguished nystagmus observed during neck stimulation from such types of preexisting nystagmus. Compere (1968) also reported a high prevalence of spontaneous nystagmus (see below) in his patients and in most of them “the nystagmus was precipitated or definitely aggravated by rotation and extension of the head and neck.”

#### 4.4. Vestibular system

None of our whiplash patients showed any spontaneous nystagmus. Spontaneous nystagmus was reported (in chronic symptomatic patients) by Oosterveld et al. (1991) in 63% of the cases and by Compere (1968) and Toglia (1976) in 29–30%, whereas Pang (1971) reported only 1 case (5%). Zenner (1991) mentioned the clinical observation of provoked (?) or gaze-evoked nystagmus in only 5% of his 100 patients.

Abnormal caloric responses were generally lacking in the present study, i.e. a substantial SE (canal paresis) was found in 4% and bilateral weakness also in 4% of our patients. Similar low percentages have been reported by others (Compere, 1968; Rubin, 1973; Oosterveld et al., 1991). Other studies mentioned canal dysfunction in 33 to 40% (Pang, 1971; Toglia, 1976; Chester, 1991). In patients with closed head injuries, canal paresis has been reported in a broad range of percentages (10–60%) unilaterally (Lange and Kornhuber, 1962; Toglia et al., 1970; Podoshin

and Fradis, 1975; Gannon et al., 1978; Tuohimaa, 1978; Vartiainen et al., 1985) and in 17% bilaterally (Lange and Kornhuber, 1962). A reduction to lower percentages (5–10%) of unilateral canal paresis has been noted in patients much later after the head trauma (Podoshin and Fradis, 1975; Tuohimaa, 1978; Vartiainen et al., 1985).

DP of rotatory responses was generally lacking in the present study. Toglia (1976) reported a significant DP of rotatory responses (i.e. “abnormal responses”, “the abnormality was basically preponderance...”) in 51%, whereas Chester (1991) mentioned “abnormalities of the vestibulo-ocular reflex” in 1 out of 9 patients tested with sinusoidal rotation.

As far as we know, vestibular hyperreactivity has not been reported in any previous study on whiplash injury. Compere (1968) specifically mentioned that “hyperactive caloric responses” were lacking. In patients with head trauma, Montandon and Dittrich (1962) found hyperreactive (their type VI) rotatory responses in almost 12%. In patients with mild head injuries, which included 48% patients who had suffered a traffic accident, Tuohimaa (1978) found a significant increase in the mean SPV (not in the nystagmus frequency, see below) during culmination of the postcaloric response measured within the first 4 days after the accident, which had normalized after 6 months; their VS test showed a significant increase in initial velocity and cumulative eye displacement only at the first examination. Kirtane et al. (1982) found a significant increase in the mean (postcaloric) nystagmus frequency in patients with head injuries (caused in a proportion of them by a car accident). VH is known to occur in multiple sclerosis (MS) (Huygen, 1983), in vestibulocerebellar dysfunction (Baloh et al., 1975), which may be also the underlying cause of the VH found in kernicterus (Huygen et al., 1993) and, furthermore, in (idiopathic) spasmodic torticollis (ST) (Huygen et al., 1989) and the HVS (Theunissen et al., 1986). The issue of VH and the HVS after whiplash injury is dealt with below. The finding of VH in ST, which we found to consist of a gain increase, may be relevant because we previously suggested that VH may represent (plastic) VOR enhancement to compensate for apparent limitation of active head movements (Huygen et al., 1989). Similar enhancement can be suggested for the present whiplash patients, because they generally had neck pain and neck stiffness and often showed limitation of neck rotation (Toglia, 1976; Oosterveld et al., 1991; Zenner, 1991). It must be noted, however, that in the whiplash patients the VOR enhancement did not involve gain enhancement (but an increase in the time constant instead, Table 2) in about half of the relevant cases (there was 1 patient with an increase in both the gain and the time constant). In patients with MS (Huygen et al., 1986) or the HVS (Theunissen, 1987) who showed VH, we observed patterns of parameter changes which were somewhat similar to those presently observed. In our previous work, we became aware of the importance of the “mental set” in



patients with the HVS (Theunissen, 1987; Theunissen et al., 1988) (see below).

#### 4.5. Respiratory system

We know of no previous reports about respiratory tests performed on whiplash patients. Almost 40% of our patients (almost 50% of the women) showed evidence of the (paroxysmal) HVS. The HVS may be placed in the context of a posttraumatic syndrome with either organic or nonorganic (“emotional”) components (or both, see below).

#### 4.6. VH, the HVS and whiplash injury

Although within the present group of patients the findings of VH and the HVS did not occur more often in combination than would have been expected on the basis of their high separate relative frequencies, it was clear (see above) that the combination of VH and the HVS could not be explained by combined false positivity.

Compared to our previous work on VH and the HVS, it appeared that the present relative frequency of the combination of VH and the HVS (7/32) was significantly lower (as shown by  $\chi^2$  tests in  $2 \times 2$  tables) than in the previously reported groups of patients selected on the basis of the HVS (20/26) (Theunissen et al., 1988) or VH (33/44) (Theunissen, 1987), in which about 3/4 of the patients showed this particular combination. It is clear therefore that the present group of whiplash patients should not be viewed as typical “hyperventilators”. VH was present in a proportion which was not significantly different from that in the HVS patients previously studied (Theunissen et al., 1988), but the proportion of subjects with the HVS was significantly lower.

Apart from possibly representing behavioural adaptation, i.e. to limited neck mobility, VH may have been caused by a lesion (dysfunction) of the vestibular commissural system (Huygen, 1983). A lesion in the brain stem may also involve the respiratory centre with its central connections. Both this centre and the VOR are under the influence of the reticular formation (the reticular activating system). The latter structure may be involved in emotional disorders and be under the influence of the “mental state” which has an important bearing on both breathing behaviour and the VOR. Folgering and Cox (1981) have shown that noradrenaline excretion is significantly increased in patients with the HVS. This would fit into a stress model in which both VH and the HVS can occur concomitantly.

#### 4.7. Further theoretical and methodological considerations

The present findings that VH and the HVS were more prominent in the women are interesting in the light of evidence that whiplash injuries occur more frequently in women than in men (Schutt and Dohan, 1968). Kahane

(1982) suggested that women are more vulnerable to whiplash injury due to the fact that “on the average, ...” they “have considerably narrower necks ... and, especially, a smaller muscle mass.”

We are fairly confident that the present (high) prevalence of abnormalities in a sample of whiplash patients overestimates the true prevalence. The true prevalence should be assessed by screening all of the subjects involved in rear-end car collisions (without contact trauma) in a certain region within a given period of time. It is clear that patient selection was not altogether avoided in the present study (or in any previous otoneurological study, as far as we know). Our observation that a lack of the symptom of dizziness was significantly associated with a short time interval between the accident and the examination suggests the existence of a selection mechanism for patients who complained (or kept complaining) about dizziness. However, we could not find any significant difference in the relative frequency of VH between the patients with and without dizziness. In other studies, the prevalence of otoneurological abnormalities could be expected to be higher than in the present study, simply because the patients had been selected on the basis of their otoneurological symptoms (and/or signs), although it seems difficult to explain why the prevalences of central and peripheral labyrinthine dysfunctions after whiplash injury were higher than after closed head injuries, including contact trauma, skull fractures and contusions (see above). Interestingly, Togliola et al. (1970) did not find any substantial difference between the prevalences of otoneurological findings after closed head trauma and whiplash injury. Central abnormalities were found only in a few cases, even after brain contusion (Lange and Kornhuber, 1962).

We reviewed the clinical records of a group of 35 consecutive routine patients referred to us (PH) whose medical history included “whiplash” (no mention was made of other trauma or disorders, use of drugs, etc.). The group comprised 25 women and 10 men. The 25:10 distribution was significantly different from the 1:1 distribution ( $p = 0.0083$  in a binomial distribution with  $p = 0.5$ ). None of our 35 routine patients showed any substantial central abnormality, although one of them showed a tendency for producing macro square wave jerks in the dark with open eyes. Seventeen out of the 25 women (significant) showed VH (68%), as well as 4 of the 10 men (40%). The referring specialist had requested respiratory function tests in only a limited number of the patients who in our opinion required such a test on account of the finding of VH and/or a history (or a clinical impression) suspicious of the HVS. In a group of 8 patients (6 tested and 2 already known to have the HVS), 7 had the HVS. Ignoring the selection procedure, we therefore knew that at least 7 out of the 35 patients had the HVS, which was a significantly high number (Table 1). At least 6 out of the 35 patients had a combination of VH and the HVS, which was



also significant. The proportion of patients with VH was fairly similar to (but slightly higher than) the corresponding proportion in men and women with VH in the present study, which also suggests that there may have been selection on symptoms in the present study. A remarkable difference was that in our routine patients, we found VH in 13 out of the 14 women (93%) whose accident had occurred more than a year before the examination, whereas VH was found in only 3 out of the 10 (30%) women (we did not know the time interval in 1 case) whose accident had occurred less than a year before the examination; the difference was significant ( $F = 0.0023$ ). As stipulated above, this suggests that our routine patients showed selective referral of cases (women) who kept complaining or started complaining later on, perhaps more so than in the present study.

We conclude that neither the findings in the present group of whiplash patients nor the findings in our routine patients provided any firm evidence of central disturbances after whiplash injury. However, this does not exclude the possibility that some form of central dysfunction was causing the features of VH and/or the HVS, as they appeared in significantly high relative frequencies both separately and in combination. Alternative explanations for these features could be sought in the hypotheses on behavioural or emotional distress.

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